Ischemic Heart Disease

- ***Pathophysiology:***
  1. ***Imbalance between coronary oxygen supply and demand***–i.e., insufficient blood flow
  2. ***Most common cause is coronary atherosclerotic disease***
  3. ***Rupture of a plaque → thrombus formation → acute occlusion***

Major Modifiable Risk Factors

- ***Elevated blood cholesterol:***
  1. LDL cholesterol is the most important
  2. Other risk factors: low HDL, high cholesterol, hypertriglyceridemia, increased total-to-HDL cholesterol ratio, and increased Lipoprotein (a)
- ***Tobacco:***
  1. Smokers have twice the risk of a nonsmoker
  2. Risk is decreased to that of a nonsmoker after 2 full years of cessation

Major Modifiable Risk Factors *(cont'd.)*

- ***Hypertension:***
  1. Risk of a cardiovascular event increases
  2. Systolic and diastolic values are of equal importance
- ***Physical Inactivity and Exercise:***
  1. Moderate exercise protects against IHD and stroke even in the ***absence*** of risk factors
  2. Can help increase HDL, control diabetes and obesity, decrease BP

Major Modifiable Risk Factors *(cont'd.)*

- ***Obesity:***
  1. Losing as little as 10-20 lbs can significantly reduce the risk of IHD if overweight
- ***Diabetes Mellitus:***
  1. Diabetes is an "IHD equivalent."
  2. Even when blood glucose is controlled, patients are still at a high risk for IHD.
  3. About **75% of patients with diabetes die** from some form of **cardiovascular disease.**

Major Uncontrollable Risk Factors

- ***Age:***
  1. Four out of five people who die of IHD are >65 years old
- ***Sex:***
  1. Males are at greater risk and develop IHD earlier
- ***Heredity:***
  1. Very important if premature disease is present

Clinical Case

A **62-year-old** **male** presents with substernal chest pain **with exertion** and is **relieved by rest**. He has been having this **on and off for 8 months**, and the last episode occurred 3 days ago while he was running to the bus. He has a history of well-controlled **diabetes** and **dyslipidemia**. The vital signs, physical exam, and ECG are all normal. An **exercise stress test** is done and shows a **2-mm ST-segment depression**.

Stable Angina

- Occurs during periods of **increased oxygen demand**: exercise and decreased supply (hypotension and anemia)
- Substernal pressure lasting 5-15 minutes with radiation to the jaw, neck, shoulders, or arms, **relieved by rest**
- Pain is usually **predictable** after a certain amount of **exertion**
- Atypical symptoms occur in the elderly or diabetics (autonomic neuropathy)

Stable Angina *(cont'd.)*

- ***Physical examination:***
  1. Typically normal
  2. May have + S_4 → stiff ventricle
- ***Laboratory studies and diagnosis:***
  1. ECG changes **during an attack**
     - Usually ST-segment depression

Exercise Stress Test

- ***Criteria for a positive stress test:***

1. >2-mm ST-segment depression

OR

2. Hypotension defined as a decrease of >10 mm Hg in systolic pressure

Exercise Stress Test

- Contraindications:
  1. Patient has suspected cardiac instability
  2. Patient is unable to walk or exercise → chemical stress test with either dipyridamole or dobutamine
  3. Baseline abnormalities (e.g. LBBB) makes interpretation difficult → nuclear stress imaging

Exercise Stress Test

- ***Validity of the test:***
  1. Affected by certain medications: β-blockers and digoxin
  2. Asymptomatic, young females have high false-positive rate
  3. Patients with known coronary artery disease have high false-negative rate

Clinical Case *(cont'd.)*

False positive test

KAPLAN MEDICAL

Other Stress Tests to Be Considered

- ***Nuclear Stress Test:***
  1. Radioactive material is used to visualize cardiac tissue perfusion
  2. Abnormal amounts of thallium are noted in areas with ↓ perfusion
  3. Overall higher sensitivity and specificity than exercise testing
  4. Bonus: not affected by baseline ECG abnormalities

Other Stress Tests to Be Considered *(cont'd.)*

- ***Dobutamine or Adenosine Stress Test:***
  1. Used in those unable to exercise
  2. Drugs are used to induce tachycardia, as if the patient was exercising
- ***Stress Echocardiogram:***
  1. Combines treadmill stress test with an echocardiogram

Invasive Testing and Therapeutics

- ***Cardiac Catheterization:***
  1. Used in patients with stable angina for diagnosis and prognosis/risk stratification
  2. Angiography is appropriate when noninvasive testing is contraindicated or inadequate
  3. Used after a positive stress test to identify patients for stent placement or bypass surgery

Treatment of Stable Angina

- Acute → sublingual nitroglycerin
- Long-term →
  1. Long-acting nitrates or β-blockers alone or in combination
  2. Aspirin (unless contraindicated)
  3. Statins
  4. Modification of risk factors should be a priority

Treatment of Dyslipidemia

- Target for hyperlipidemic patients **with CAD:**
  1. LDL → <100 mg/dL
  2. HDL → >40 mg/dL
  3. Triglycerides → < 150 mg/dL
- Target for **very high-risk** hyperlipidemic patients:

Treatment of Dyslipidemia *(cont'd.)*

- Criteria for being a "very high-risk" patient:
  1. Those with established coronary artery disease ***plus*** ***any one of the following:***
     - Multiple major risk factors
     - Metabolic syndrome-associated risk factors
     - Acute coronary syndrome

Treatment of Dyslipidemia *(cont'd.)*

- First-line treatment → **statins**
- If the patient is intolerant to a prescribed statin → decrease the dose and change to another statin

CABG and PCI

- ***Coronary Artery Bypass Grafting (CABG)***
  1. Useful in either:
     - Left main coronary disease
     - Three-vessel disease and LV dysfunction
     - Symptoms despite medical therapy or severe side effects from therapy
  2. More efficacious in those with diabetes
  3. More beneficial but risky in those with low ejection fractions
- ***Percutaneous Coronary Intervention (PCI)***
  1. Stent placement is now the standard of care.

Etiology

- Etiology may be benign *or* life-threatening
  1. Evaluation should focus on **excluding the most serious conditions first**

History Taking

- **History of chest pain** is **more useful than the physical exam**
  1. Duration
  2. Quality
  3. Location
  4. Radiation
  5. Frequency
  6. Alleviating factors
  7. Precipitating factors (**especially exercise**)

Clinical Pearls to Remember

- Stable angina and acute coronary syndromes
  1. Character: "tightness," "heaviness," or "pressure"
- Inferior wall ischemia
  1. May present with vagal reflexes: bradycardia, hypotension, and/or dizziness/fainting
- Less likely to be ischemia/infarction
  1. "Sharp" or "knife-like" pain that is easily pin-pointed
  2. Pain is reproduced on palpation or changes in position

Clinical Pearls to Remember *(cont'd.)*

- True infarction
  1. Pain lasts >20-30 minutes in duration
- Transient ischemia (angina) or esophageal spasm
  1. **Relieved** by nitroglycerin
- Gastroesophageal reflux disease (GERD)
  1. Can **worsen** with nitroglycerin

Physical Exam

- "Initial impression" is extremely important
  1. Diaphoresis, tachypnea, and/or anxiousness should alert the physician to a potentially **life-threatening process**
  2. Tachycardia and tachypnea (usually nonspecific) are **always present with pulmonary embolism.**

Physical Exam *(cont'd.)*

- Check for abnormal heart sounds or new onset murmurs. Associations to bear in mind:
  1. Wide split of S_2 → RBBB (right bundle branch block) or right ventricular infarction
  2. New paradoxic split of S_2 → LBBB (left bundle branch block), anterior, or lateral infarction
  3. New S_4 → angina or infarction
  4. S_3 → underlying CHF
  5. New onset aortic regurgitation → 50% of patients with aortic dissection
  6. New onset mitral regurgitation → papillary muscle dysfunction

Testing

- ***All patients with chest pain should have a 12-lead ECG!!! This is the single most important test for evaluation of cause!!!***
- ***An ECG consistent with myocardial infarction (MI) is the only test required to select patients for emergency reperfusion!!!***

Acute Myocardial Infarction

KAPLAN MEDICAL

Testing *(cont'd.)*

- ***Cardiac Biomarkers***
  1. Cardiac troponins and CK-MB are the two most important markers
     - **CK-MB Isozyme** → present **4-6 hours after the onset of ischemia**, peaks in 12-24 hours, and normalizes in 3-4 days. Predicts **early re-infarction**. Often runs in serials every 6-12 hours

Testing *(cont'd.)*

- ***Cardiac Biomarkers (cont'd.)***
  1. Cardiac troponins and CK-MB are the two most important markers *(cont'd.)*
     - CK-MB Subforms → CK-MB1 found in plasma, CK-MB2 found in myocardial tissue.
       - CK-MB2 >1 U/L and a subform ratio of 1.5 is 96% sensitive and 94% specific

Testing *(cont'd.)*

- ***Cardiac Biomarkers (cont'd.)***
  1. Cardiac troponins and CK-MB *(cont'd.)*
     - Cardiac Troponins → ***preferred markers for diagnosis of myocardial injury.*** Troponin is also elevated in renal disease, polymyositis/dermatomyositis
       - Normal CK-MB + ↑ troponin = ***minor myocardial damage***
       - ↑ CK-MB + ↑ troponin = ***acute myocardial infarction***
       - Remain elevated for weeks → good ***late markers of myocardial infarction***

Noncardiovascular Disorders	Differentiating Features
Costochondritis	Pain exacerbated with inspiration; ***reproduced with chest wall palpation***
GERD	***Acid reflux***; relief with antacids
Peptic ulcer	Epigastric pain
Gallbladder disease	***RUQ*** abdominal pain and tenderness

Cardiovascular Disorders	Differentiating Features
Myocardial Infarction	Pain more ***severe***, usually ***>20 minutes in duration***
Aortic Stenosis	Typical ***systolic ejection murmur***
Myocarditis	Pain is ***vague and mild***
Pericarditis	Pain is ***sharp, worse with lying down, relieved by sitting up***
Dissecting aortic aneurysm	Pain is ***sharp, tearing***, usually in the ***back***
Mitral valve prolapse	***Transient*** pain, ***midsystolic click, young female*** with ***no risks***

Pulmonary Disorders	Differentiating Features
Pulmonary embolus/infarction	***Tachypnea***, ***dyspnea***, cough, pleuritic pain, hemoptysis
Pneumothorax	***Sudden*** onset of ***pain + dyspnea***

Causes of Chest Pain

- ***Aortic Dissection:***
  1. Severe, sharp, tearing chest pain radiating to the back
  2. Loss of pulses and/or aortic insufficiency
  3. Widened mediastinum on chest x-ray

Causes of Chest Pain

- ***Aortic Dissection:***
  1. Severe, sharp, tearing chest pain radiating to the back
  2. Loss of pulses and/or aortic insufficiency
  3. Widened mediastinum on chest x-ray
  4. MI may occur if dissection involves the coronary artery
  5. Diagnosis is confirmed with MRI, CT scan, TEE (transesophageal echocardiogram), or aortography

Causes of Chest Pain *(cont'd.)*

- ***Pulmonary Embolism:***
  1. Sudden onset dyspnea, tachypnea, and hypoxemia is prominent
  2. Pleuritic pain, especially with pulmonary infarction
  3. ECG is usually nonspecific
  4. Diagnosis is confirmed with spiral chest CT scan, lung scan, or pulmonary angiogram.

Causes of Chest Pain *(cont'd.)*

- ***Pericarditis:***
  1. May be preceded by a viral illness
  2. Pain is sharp, positional, pleuritic, and relieved by leaning forward
  3. Pericardial friction rub
  4. Diffuse ST elevation without Q-waves
  5. Creatinine kinase usually normal
  6. Responds to NSAIDs

Causes of Chest Pain *(cont'd.)*

- ***Myocarditis:***
  1. May be preceded by a viral illness (most common etiology in the U.S.). Highly associated with ***Coxsackie B virus***. Most common worldwide cause is diphtheria. Most common cause in South Americans is ***Chagas disease***
  2. Pain is usually vague and mild.
  3. Total CK and CK-MB are often elevated
  4. +/- conduction abnormalities, and sometimes Q-waves

Causes of Chest Pain *(cont'd.)*

- ***Pneumothorax:***
  1. Abrupt onset with sharp pleuritic chest pain and dyspnea; breath sounds absent
  2. Confirm with chest x-ray
- ***Pleuritis:***
  1. Sharp pain increases with inspiration
  2. Friction rub or dullness

Acute Coronary Syndrome

Acute Coronary Syndrome is used to describe a range of thrombotic syndromes. What are they?

1. Unstable angina

2. Non-ST elevation myocardial infarction (NSTEMI)

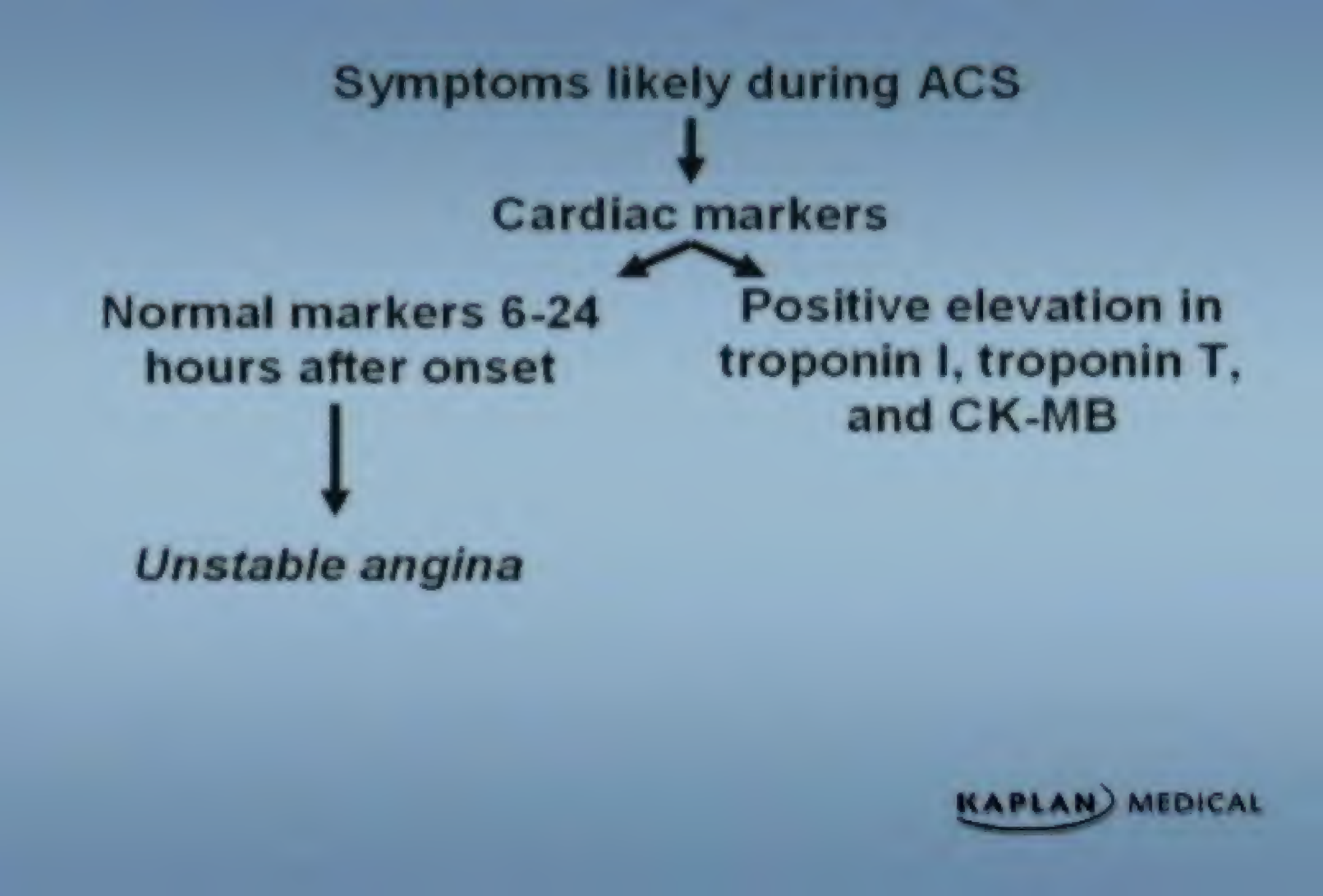
Acute Coronary Syndrome: Overview

- The initial presentation and management of unstable angina, NSTEMI, and STEMI are very much the same
- Important to distinguish ACS from stable angina

Location of Pain Associated With ACS

These images were reproduced from Wikipedia,
http://www.wikipedia.com

Unstable Angina and NSTEMI


Symptoms likely during ACS
Cardiac markers
Normal markers 6-24 hours after onset
Positive elevation in troponin I, troponin T, and CK-MB
Unstable angina
KAPLAN MEDICAL

Unstable Angina and NSTEMI *(cont'd.)*

- ***High-risk features:***
  1. Repetitive or prolonged chest pain (>10 minutes)
  2. Elevated cardiac markers
  3. Persistent ECG changes
  4. Hemodynamic instability
  5. Sustained ventricular tachycardia
  6. Syncope
  7. Left ventricular ejection fraction less than 40%
  8. Prior PTCA or CABG
  9. Diabetes
  10. Chronic renal disease

Unstable Angina and NSTEMI *(cont'd.)*

- ***Management:***
  1. Thrombolytic therapy is NOT effective!! Infarcted artery is NOT occluded in 60-85% of cases

Unstable Angina and NSTEMI *(cont'd.)*

- ***Management (cont'd.):***
  3. Antiplatelet therapy: aspirin + clopidogrel ***unless contraindications exist***
  4. Antithrombin therapy: unfractionated heparin or subcutaneous enoxaparin for 48-72 hours or until angiography is performed
  5. Glycoprotein IIb/IIIa inhibitors (tirofiban or eptifibatide): especially in high-risk patients
  6. β-blockers unless contraindicated
  7. Intravenous nitroglycerin for refractory pain

Unstable Angina and NSTEMI *(cont'd.)*

- ***Invasive Management:***
  1. Early coronary angiography (within 48 hours) and revascularization is recommended in patients with severe comorbidity

STEMI
(Formerly Known as Q-wave MI)

STEMI

- ***Symptoms:***
  1. Substernal pain/pressure with radiation to the jaw, neck, shoulders, or arms, lasting >20 minutes and not relieved completely with nitroglycerin
  2. Dizziness, nausea/vomiting, diaphoresis, or shortness of breath
  3. Elderly or diabetics often atypical
  4. Physical findings are variable depending on infarction area and size

STEMI *(cont'd.)*

- ***Diagnosis:***
  1. Clinical symptoms

     Plus the following ECG Changes:

     - Persistent ST-segment elevation of >1 mm in two contiguous leads

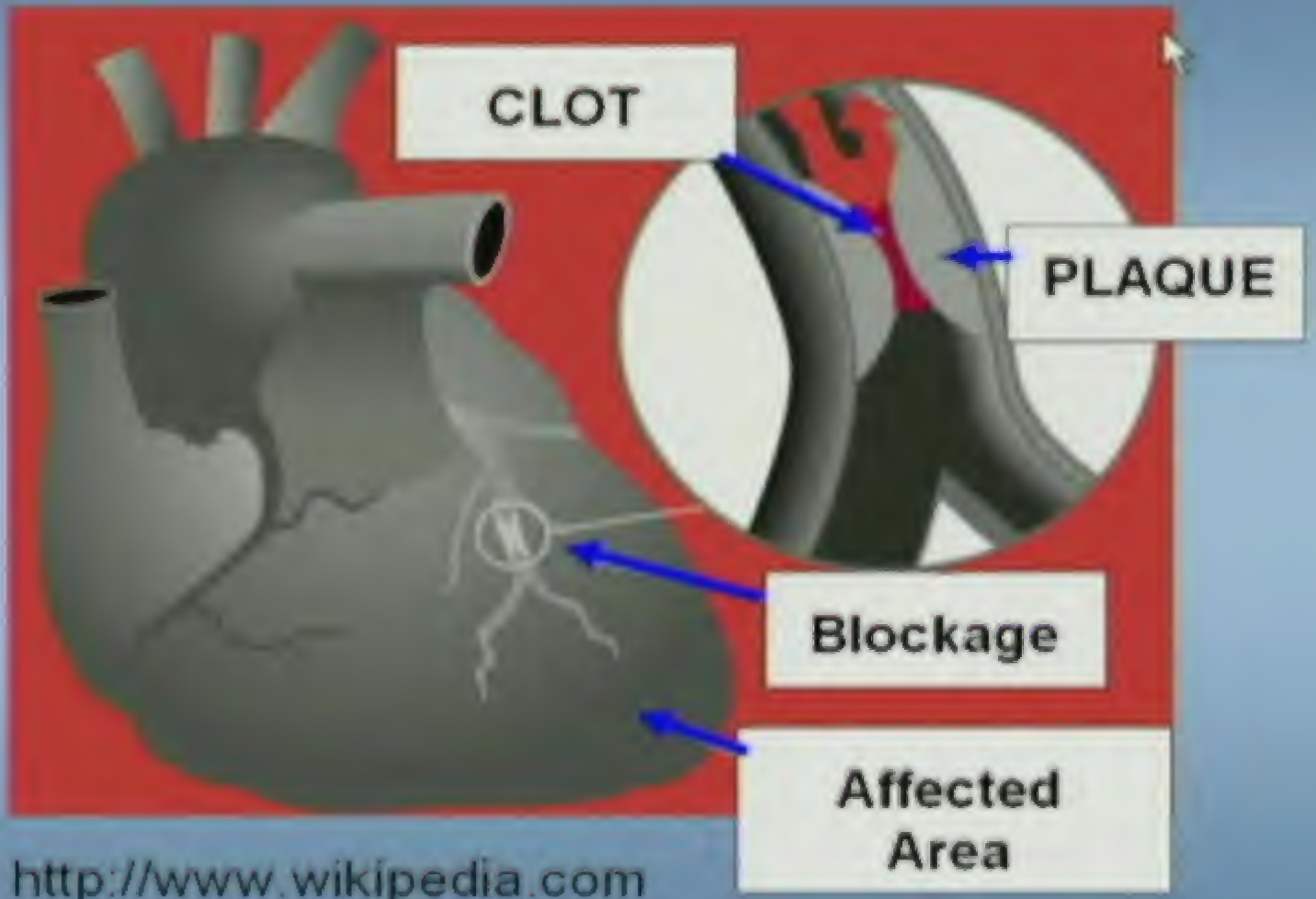
STEMI *(cont'd.)*

This means that you do not initially need elevated biochemical markers for the diagnosis of STEMI!

Area Infarcted	ECG Changes	Artery
Inferior	II, III, aVF	RCA
Anteroseptal	V_1-V_3	LAD
Anterior	V_2-V_4	LAD
Lateral	I, aVL, V_4, V_5, and V_6	LAD or circumflex
Posterior	V_1-V_2: Tall, broad initial R-wave; ST depression; tall, upright T-wave; usually occurs in association with inferior or lateral MI	

Table I-6-6: ECG Evolution of MI

ECG Change	Onset	Disappears
Hyperacute T-waves (tall, peaked T-waves in leads facing the infarct	Immediate	6-24 hours
ST segment elevation	Immediate	1-6 weeks
Q-waves >0.04 seconds	One-several days	Years to never
T-wave inversion	6-24 hours	Months to years


CLOT
PLAQUE
Blockage
Affected Area
http://www.wikipedia.com

Emergent Reperfusion Therapy *(cont'd.)*

- ***Choice between PCI versus thrombolysis (cont'd.)***
  3. Where PCI is delayed or not available, reperfusion with thrombolysis is best
  4. Greatest benefit is to those with ***anterior infarction***, ST-elevation, or bundle branch block and symptoms for <12 hours
  5. Streptokinase and alteplase → rapid IV infusion
  6. Reteplase and tenecteplase → rapid bolus injection

Emergent Reperfusion Therapy *(cont'd.)*

- ***The bottom line:***

1. Consider thrombolytic therapy as an alternative to primary PCI in suitable candidates with:
   - ST-elevation MI (>1 mm ST-elevation in two contiguous leads)
   - New LBBB (left bundle branch block)

Contraindications to Thrombolytics

- ***Absolute Contraindications:***
  1. Active bleeding or bleeding diathesis
  2. Significant closed head or facial trauma within 3 months
  3. Suspected aortic dissection
  4. Prior intracranial hemorrhage
  5. Ischemic stroke within three months

Contraindications to Thrombolytics *(cont'd.)*

- ***Relative Contraindications:***
  1. Recent major surgery (<3 weeks)
  2. Traumatic or prolonged cardiopulmonary resuscitation
  3. Recent (within 4 weeks) internal bleeding
  4. Active peptic ulcer
  5. Severe, poorly controlled hypertension
  6. Ischemic stroke <3 months

Late Presentation: 12 Hours After Onset of Symptoms

- Reperfusion and PCI are not indicated in stable patients
- CABG may be considered in those with good anatomy
- CABG is also considered in those with cardiogenic shock or in association with mechanical repair

Adjuvant Therapy Used With Reperfusion

- ***Antiplatelet therapy:***
  1. Aspirin → all patients unless contraindicated
  2. Clopidogrel → all patients undergoing PCI with stent placement and those undergoing fibrinolytic treatment

Adjuvant Therapy Used With Reperfusion *(cont'd.)*

- ***Glycoprotein IIb/IIIa Inhibitors:***
  1. Primary PCI → early use has greatest benefit
- ***Emergency CABG:***
  1. Failed PCI
  2. Persistent or recurrent ischemia refractory to medical therapy

Other Testing in ACS

- ***Exercise ECG Testing (Stress Test):***
  1. Submaximal testing 4-7 days after infarction
  2. Test 3-6 weeks post-infarction maximal
  3. Assesses prognosis and those with reversible ischemic who may need an angiogram and/or CABG

- ***Myocardial Perfusion Imaging:***

Discharge Medications After ACS

- Aspirin → 75-325 mg/day
- Clopidogrel → in intolerant to aspirin or as an alternative for 9-12 months
- β-blocker → indefinitely
- ACE inhibitors → added for CHF or left ventricular dysfunction (EF <40%) is present
- Statins → should be started in the hospital
- Nitrates → Short-acting nitrates as a rule. Long-acting only if chest pain is persistent.

Discharge Medications After ACS *(cont'd.)*

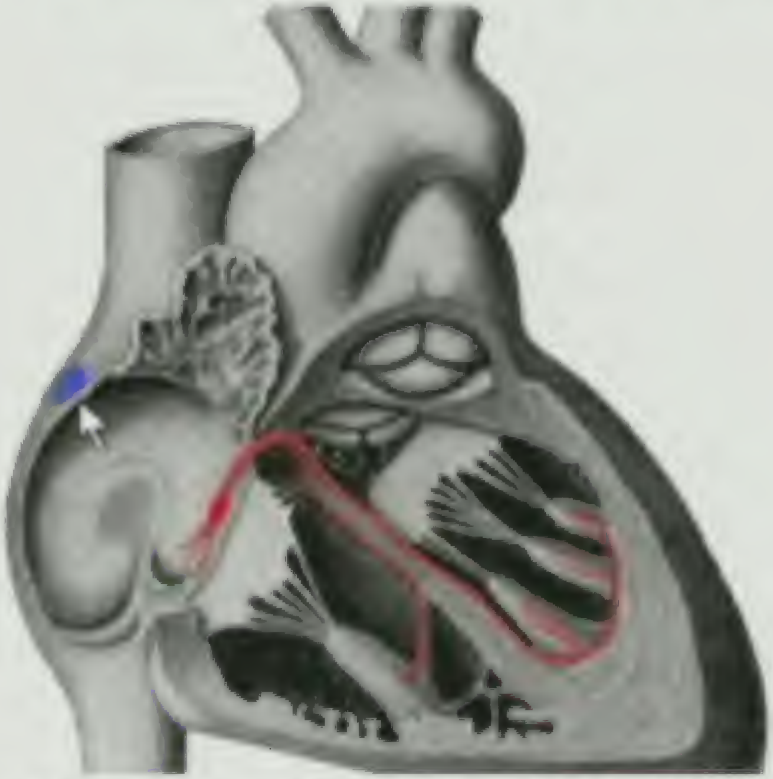
Pneumonic	*Significance*
A	Aspirin and anti-anginals
B	β-blockers and blood pressure
C	Cholesterol and cigarettes
D	Diet and diabetes
E	Education and exercise

Complications of ACS

Electric Disturbances

- ***Dysrhythmias:***
  1. Bradycardia
  2. Premature beats
  3. Tachyarrhythmias → supraventricular or ventricular
- ***Conduction Abnormalities:***
  1. Atrioventricular nodal

Normal Conduction

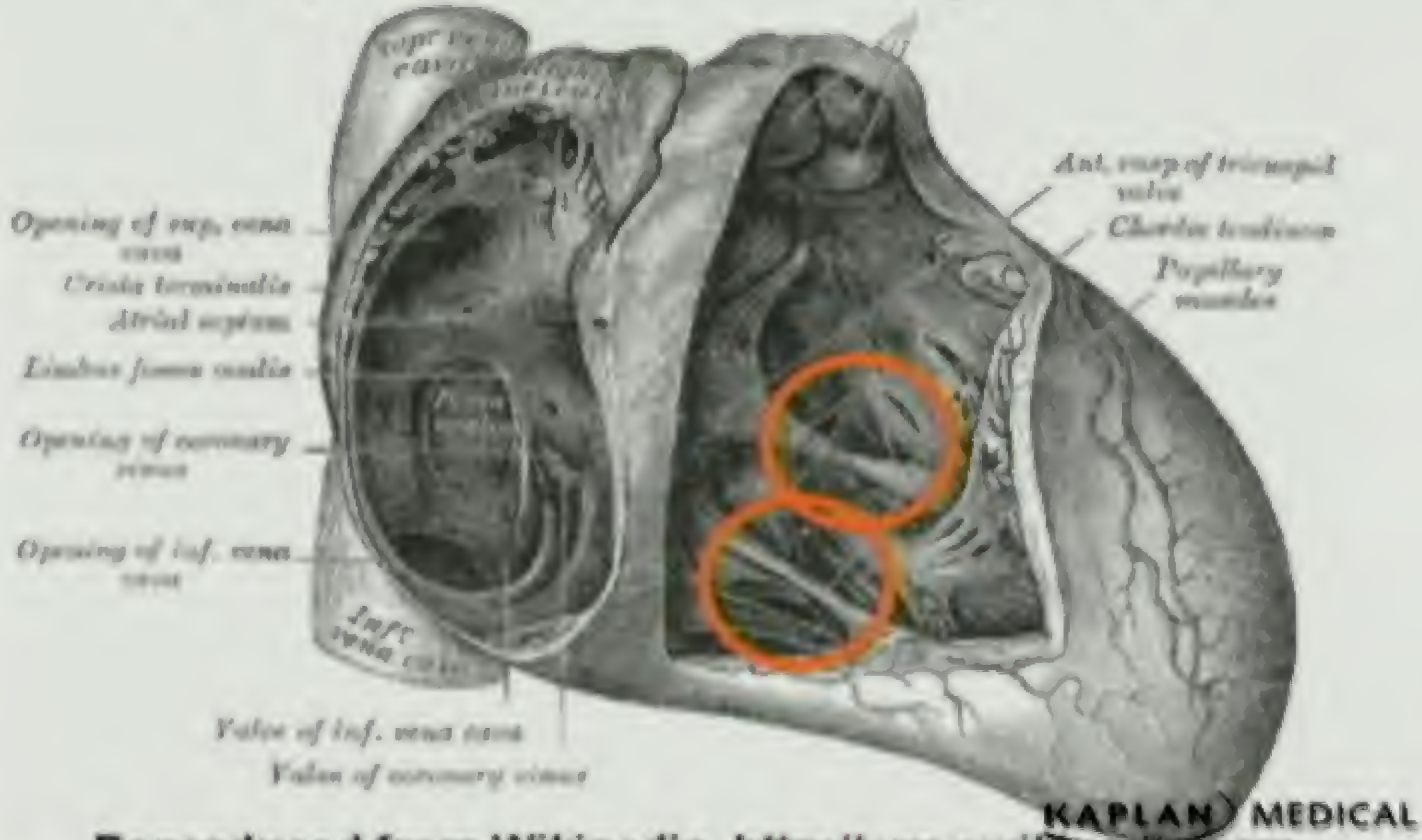


This image was reproduced from Wikipedia,
http://www.wikipedia.com

Pump Dysfunction

- Contractile dysfunction
- Mechanical disruption
- Electromechanical dissociation

Papillary Muscle Rupture



KAPLAN MEDICAL

Reproduced from Wikipedia, http://www.wikipedia.com

ACS Complications *(cont'd.)*

- Ischemia
- Pericarditis → Dressler syndrome
  1. Treat with NSAIDs, aspirin, +/- steroids
- Thromboembolic → mural thrombi or deep venous thrombosis

Prinzmetal Angina

- Secondary to sudden coronary ***vasospasm***
- Associated with ***ST-segment elevation***
- Occurs at ***rest***
- Patients often ***awaken from sleep*** with severe chest pain
- Occurs more often in ***females*** and those with a history of migraine
- Stress testing and angiography are normal
- Definitive diagnosis → ***ergonovine during angiography*** → triggers vasospasm → treatment with ***calcium-channel blockers or nitrates***

Mitral Stenosis: Etiology

- ***Most common lesion due to rheumatic fever***
- Rarely due to a genetic defect
- 2/3 of patients are female

Mitral Stenosis: Symptoms

- Dyspnea
- Orthopnea/paroxysmal nocturnal dyspnea
- Fatigue
- Wasting
- Hemoptysis (ruptured vessels)
- Systemic embolism (stagnation of left atrial blood)
- Hoarseness (enlarged left atrium on the recurrent laryngeal nerve)
- Right-sided heart failure: hepatomegaly, ascites, peripheral edema

Mitral Stenosis: Signs

- Atrial fibrillation
- Rales
- Decreased pulse pressure
- Loud S_1
- ***Opening snap following S_2***
- ***Diastolic rumble (low-pitched, apical murmur)***
- Sternal lift (due to enlarged right ventricle)

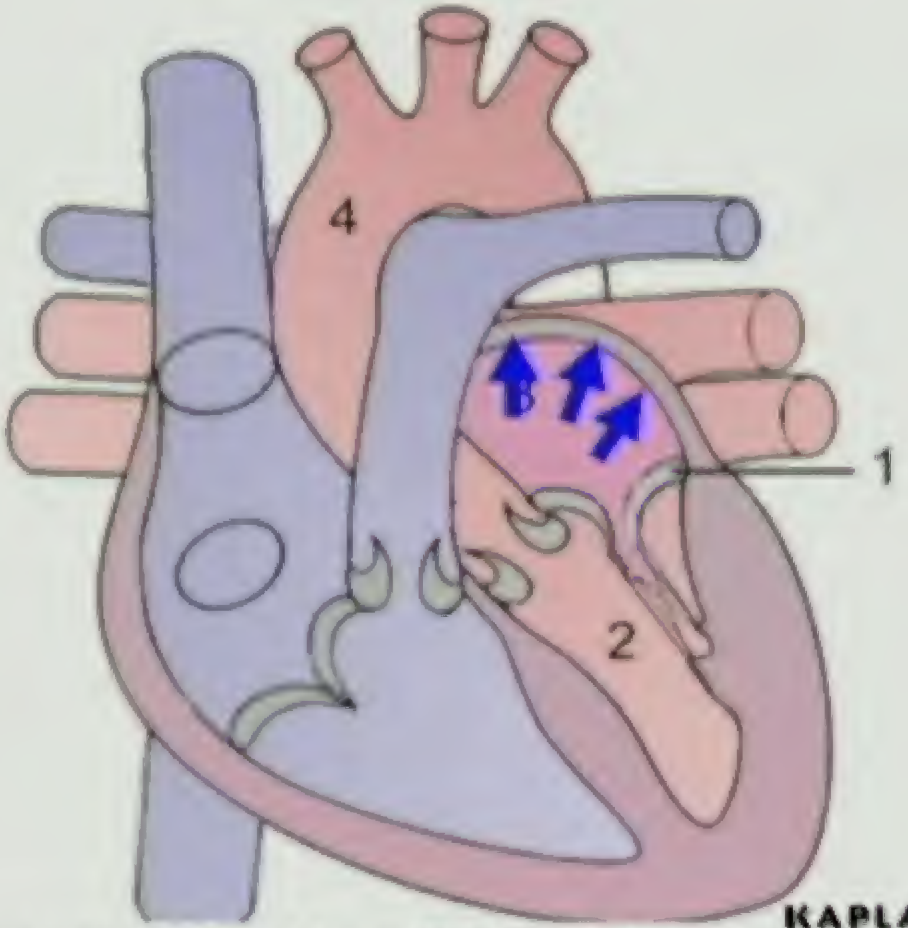
Mitral Stenosis: Diagnosis

- ***EKG:***
  1. May show right ventricular hypertrophy
  2. May show right and left atrial abnormalities
  3. +/- A-fib
- ***Chest x-ray:***
  1. Large left atrium, pulmonary hypertension, large pulmonary artery are all possible
- ***Echocardiography:***
  1. Thickened mitral valve leaflets and decreased excursion

Mitral Stenosis: Treatment

- ***Medical therapy:***
  1. Diuretics and salt-restricted diet
  2. Digitalis and anticoagulants in A-fib
- ***Surgical therapy:***
  1. Balloon valvuloplasty
  2. Mitral commissurotomy or valve replacement
  3. Pulmonary hypertension does not contraindicate surgery

Mitral Regurgitation



KAPLAN MEDICAL

This image was reproduced from Wikipedia, http://www.wikipedia.com

Mitral Regurgitation: Etiology

- Due to abnormalities in the mitral valve leaflets, annulus, and cordae tendinae
  1. Common cause is dilation of the left ventricle
  2. Men > women

Mitral Regurgitation: Diagnosis

- ***Physical Signs:***
  1. Hyperdynamic, displaced apical impulse
  2. Carotid upstroke decreased but brisk
  3. Holosystolic apical murmur radiating to the axilla +/- thrill
  4. S_3 usually heard with a soft S_1 and widely split S_2
  5. Distended neck veins

Mitral Regurgitation: Diagnosis *(cont'd.)*

- *EKG* → left-ventricular hypertrophy and left atrial enlargement
- *Chest x-ray* → cardiac enlargement with vascular congestion
- *Echocardiography* →left atrial and/or left ventricular dilation

Mitral Regurgitation: Treatment

- ***Medical Therapy:***
  1. Increase cardiac output and relieve pulmonary congestion
     - Arteriolar vasodilators
     - Digitalis
     - Diuretics
     - Anticoagulants
- ***Surgery:***
  1. Guidelines for selecting patients:
     - With significantly limiting symptoms and severe regurgitation
     - Symptoms persist despite optimal medical therapy

Mitral Valve Prolapse

- **Most common congenital valvular lesion**
- More common in **females** and those with **connective tissue disease**
  1. Marfan syndrome
- ***Presentation:***
  1. Usually asymptomatic unless arrhythmias occur: chest pain, lightheadedness, palpitations, and syncope

Mitral Valve Prolapse *(cont'd.)*

- ***Auscultation:***
  1. **Mid-to-late systolic click** and late systolic murmur heard best at the apex
  2. Worsens with Valsalva maneuver
  3. Improves with squatting
- ***Complications:***
  1. Serious arrhythmias and sudden death
  2. CHF
  3. Bacterial endocarditis
  4. Calcifications of the valve
  5. TIAs

Mitral Valve Prolapse *(cont'd.)*

- ***Labs:***
  1. 2-D Echo → systolic displacement of the mitral leaflets
- ***Treatment:***
  1. Medical management → endocarditis prophylaxis if a murmur is present, β-blocker for chest pain/arrhythmia

Aortic Stenosis

Aortic Stenosis: Pathophysiology

- Calcification and degeneration of a congenitally normal valve, most commonly in the elderly
- Calcification and fibrosis of congenital bicuspid valve
- Rheumatic valvular disease

Aortic Stenosis: Symptoms

- Angina, syncope, and dyspnea
- Pulsus tardus et parvus
- Carotid thrill
- Systolic ejection murmur heard best at right sternal border, with a thrill, harsh quality, and radiates to the carotids
- S_4 gallop
- A_2 decreased, S_2 single or paradoxic split

Aortic Stenosis *(cont'd.)*

- ***Diagnosis:***
  1. ECG: LV hypertrophy
  2. Chest x-ray: calcifications, cardiomegaly, pulmonary congestion
  3. Echocardiography: thick aortic valve leaflets with decreased excursion and LVH
- ***Treatment:***
  1. Patients must receive endocarditis prophylaxis
  2. Surgery when valve area is ↓ <0.8 cm^2
  3. Generally, if patients have symptoms, they need surgery
  4. Balloon valvuloplasty in those who cannot tolerate surgery

Aortic Regurgitation

Aortic Regurgitation

- Most common cause is *systemic hypertension*
- Other causes include:
  1. Infectious endocarditis
  2. Syphilis
  3. Ankylosing spondylitis
  4. Marfan syndrome
  5. Aortic dissection

Aortic Regurgitation *(cont'd.)*

- ***Clinical findings:***
  1. Dyspnea most common
  2. Diastolic-decrescendo murmur most common
  3. Systolic flow murmur
  4. Duroziez sign: systolic/diastolic thrill or murmur over the femoral arteries
  5. S_3 in early left ventricular decompensation

Aortic Regurgitation *(cont'd.)*

- ***Diagnosis:***
  1. ECG: LV hypertrophy
  2. Chest x-ray: LV or aortic dilation
  3. Echocardiography: Dilated LV and aorta; left-ventricular volume overload; fluttering of anterior mitral leaflet
- ***Treatment:***
  1. Endocarditis prophylaxis
  2. Salt restriction, diuretics, afterload reduction
  3. Aortic valve replacement when symptoms are severe or ejection fraction plummets

KAPLAN MEDICAL

Myocardial and Pericardial Disease

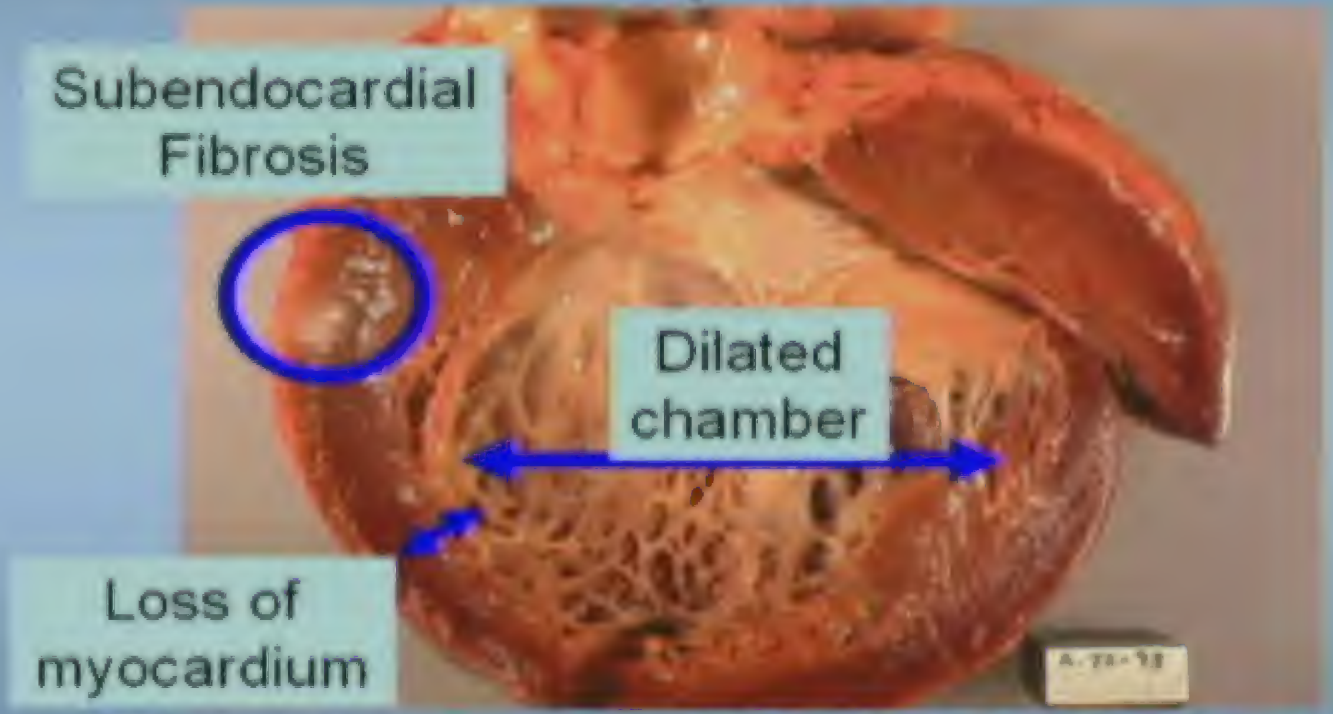
Cardiomyopathy: Overview

Subtype	***Characteristics***
Dilated	Biventricular dilation
Hypertrophic	Marked hypertrophy of the left ventricle +/- right ventricle, disproportionate hypertrophy of the septum
Restrictive	Reduced compliance, due to infiltration of the myocardium
Obliterative	Thickened endocardium or mural thrombi, or both, act as space-occupying lesions

Dilated Cardiomyopathy

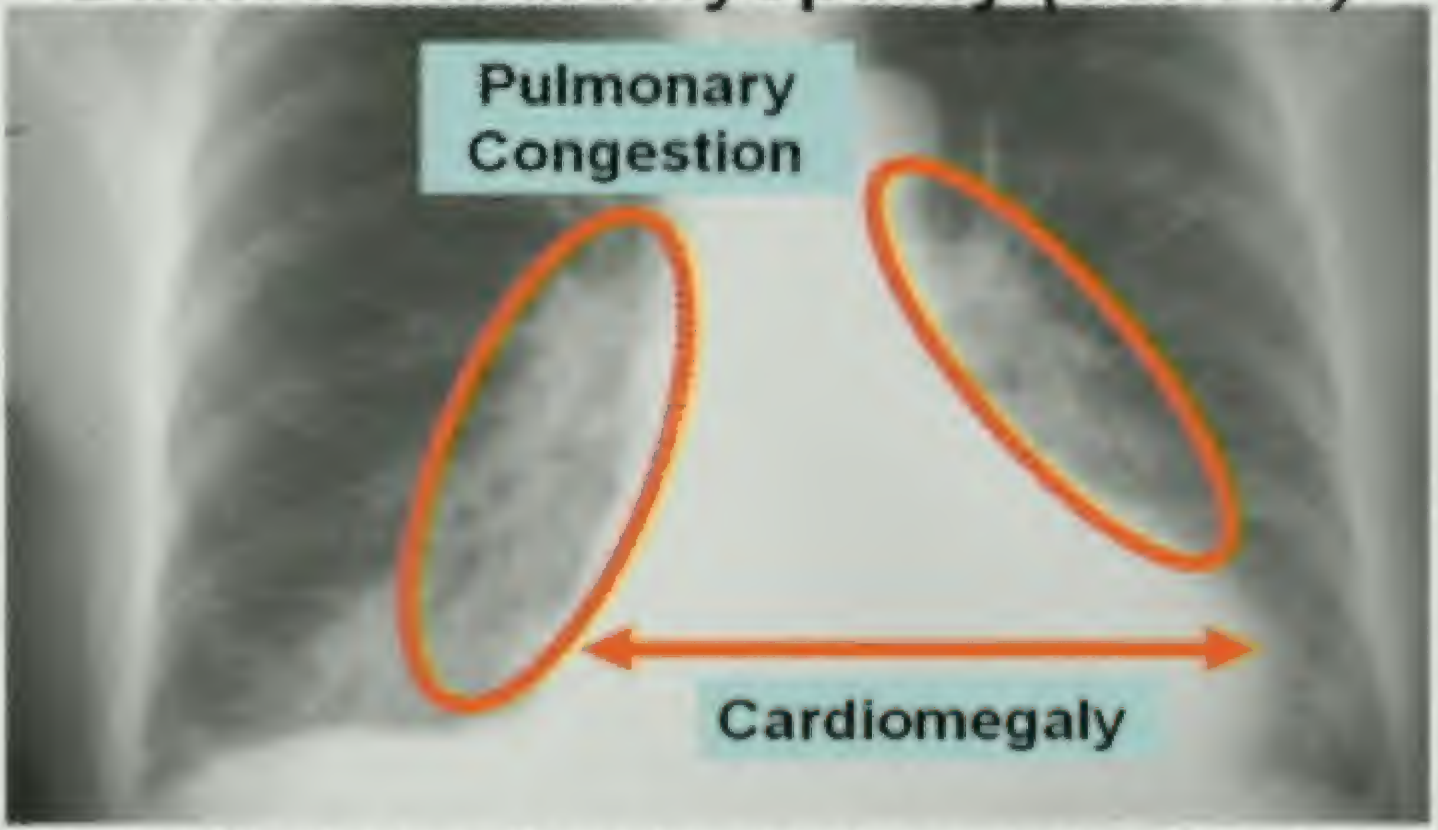
- Diminished contractility
- Usually both ventricles
- ***Most common indication for heart transplant***
- ***Etiology:***
  1. Ischemic → ***most common cause***
  2. Alcoholism → ***2nd most common cause***
  3. Peripartum
  4. Post-myocarditis
  5. Toxins (lead, cobalt, arsenic)
  6. Doxorubicin hydrochloride, cyclophosphamide, vincristine

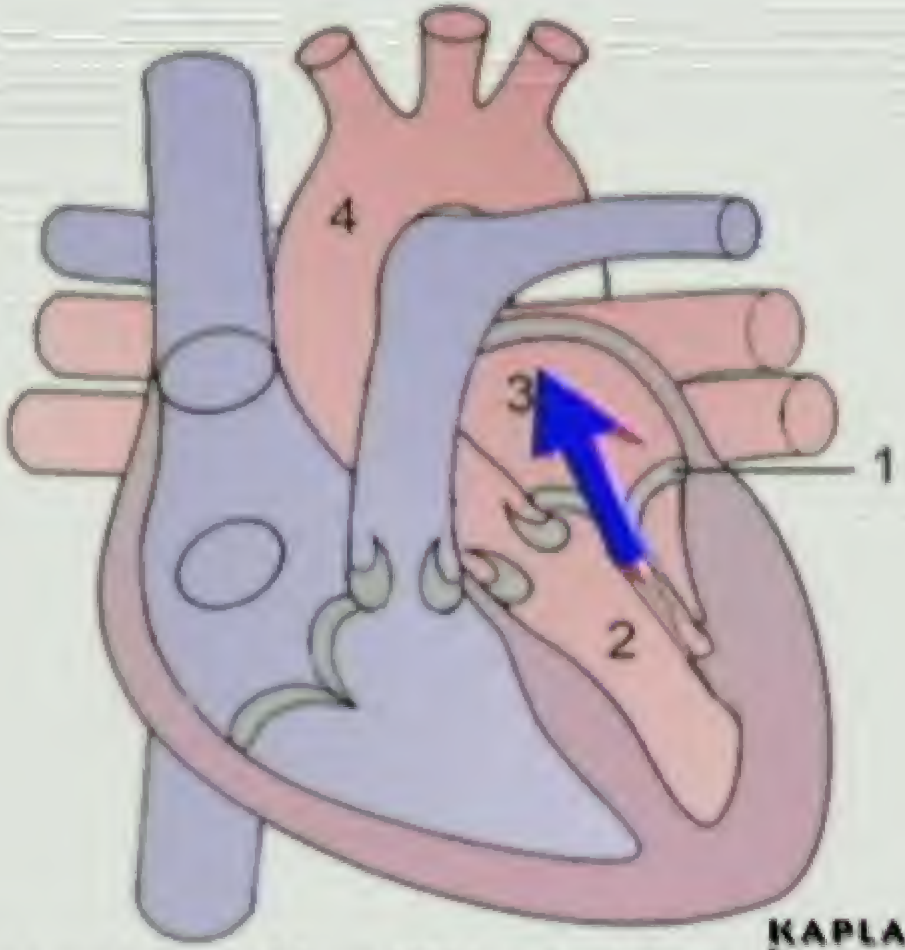
Dilated Cardiomyopathy: Idiopathic



This image was reproduced from the Public Health Image Library, http://phil.cdc.gov.

Dilated Cardiomyopathy *(cont'd.)*





KAPLAN MEDICAL

This image was reproduced from Wikipedia, http://www.wikipedia.com

Factors That Increase Obstruction

Mechanism	*Physiologic or Pharmacologic Factors*
↑ Contractility	• Cardiac glycosides • β-agonists • Tachycardia • Premature beats
↓ Preload	• Valsalva or standing • ↓ Intravascular volume • Nitrates and vasodilators • Tachycardia
↓ Afterload	• Hypovolemia • Nitrates and vasodilators

Hypertrophic Obstructive Cardiomyopathy *(cont'd.)*

- ***Clinical Manifestations:***
  1. Dyspnea
  2. Angina/palpitations/presyncope-syncope
  3. Palpable S_4 gallop, systolic murmur + thrill, mitral regurgitation murmur
  4. Sudden death can be the first manifestation

Hypertrophic Obstructive Cardiomyopathy *(cont'd.)*

- ***Diagnosis:***
  1. ECG → Left axis deviation, pseudo Q-waves (V_1-V_3), ventricular arrhythmias
  2. Echo → ***Mainstay of diagnosis***; hypertrophy, systolic anterior motion of mitral valve, midsystolic closure of aortic valve
- ***Treatment:***
  1. β-blockers
  2. Calcium channel blockers
  3. Disopyramide on occasion
  4. Surgery for severe cases

Restrictive Cardiomyopathy

- Least common cardiomyopathy
- Characterized by ***rigid, noncompliant ventricular walls***

- ***Etiology:***
  1. Infiltrative:
     - Sarcoidosis, amyloidosis, hemochromatosis, neoplasia

Restrictive Cardiomyopathy *(cont'd.)*

- ***Clinical Manifestations:***
  1. Dyspnea, exercise intolerance, and weakness
  2. Edema, elevated JVP, hepatomegaly, ascites, S_4 and S_3 gallop, Kussmaul sign

Restrictive Cardiomyopathy *(cont'd.)*

- ***Diagnosis:***
  1. X-ray → mild cardiomegaly, pulmonary congestion
  2. ECG → low voltage, conduction disturbances, Q-waves
  3. Echo → rigid myocardium, characteristic seen in amyloidosis with thickening of all cardiac structures
- ***Treatment:***
  1. No good treatment

Acute Pericarditis

- ***Etiology:***
  1. Idiopathic
  2. Infections
  3. Vasculitis
  4. Disorders of metabolism
  5. Neoplasms
  6. Trauma

Acute Pericarditis *(cont'd.)*

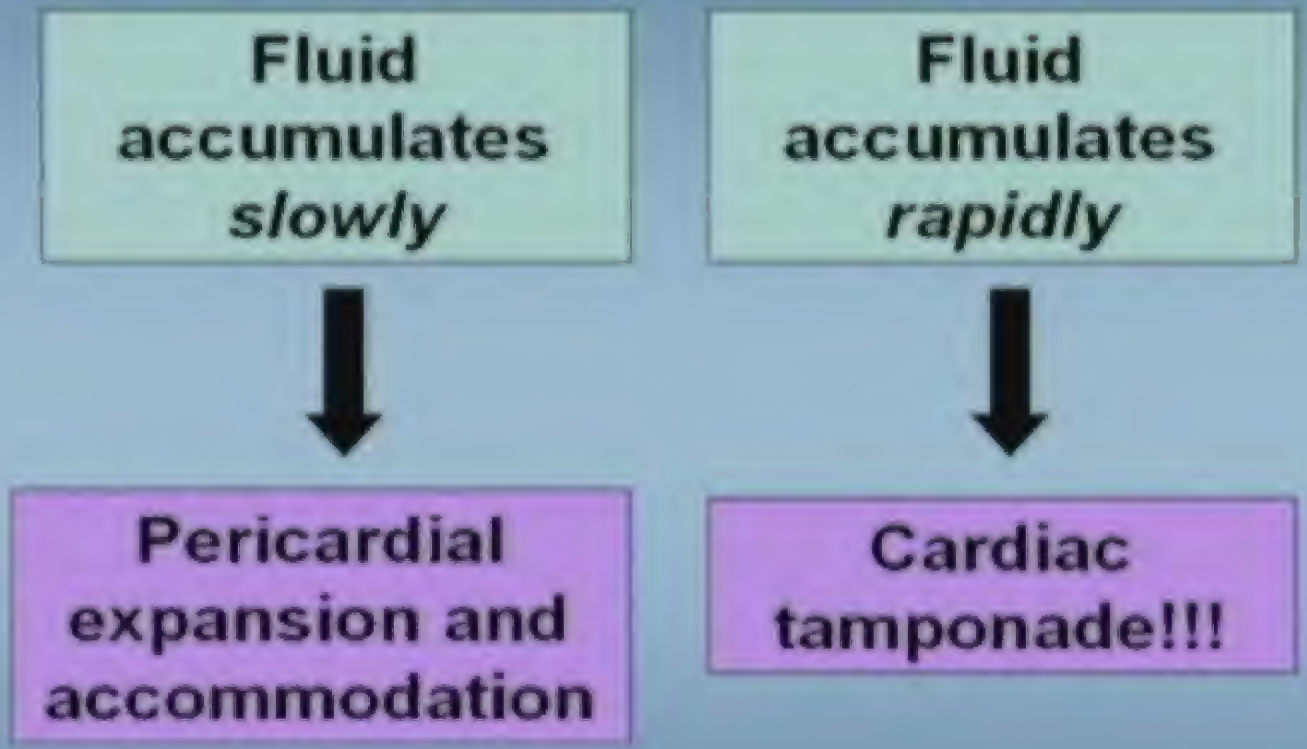
- ***Clinical presentation:***
  1. Substernal-left sternal chest pain worsened with recumbency, coughing, and deep inspiration
  2. Relieved by sitting up or leaning forward
  3. Pericardial friction rub
- ***Diagnosis:***
  1. ECG → diffuse ST-segment elevation with upright T-waves

Acute Pericarditis *(cont'd.)*

- ***Differential Diagnosis:***
  1. Acute MI → pericarditis has characteristic ST-segment elevation, **absence of reciprocal leads,** and **no Q-waves**
- ***Treatment:***

Pericardial Effusion

Character	***Etiology***
Transudate	CHF, fluid overload, hypoproteinemia
Exudate	Pericardial injury
Sero-sanguinous	Tuberculosis, neoplasm
Frank blood	Aortic aneurysm, dissection, blunt/penetrating trauma, bleeding 2° to coagulation defects, myocardial rupture post-MI



Pericardial Effusion *(cont'd.)*

- ***Diagnosis:***
  1. Echocardiography →
     - Echo-free pericardial space between the posterior pericardium and posterior left ventricular epicardium
     - In large effusions, the heart may swing freely, and may be associated with electric alternans
  2. Chest x-ray → "water-bottle" configuration of the cardiac silhouette
- ***Treatment:***

Cardiac Tamponade

- Medical emergency!!!
- Effusion develops rapidly → compression of the heart → rapid heart failure
- ***Etiology:***
  1. Neoplasia
  2. Idiopathic (usually viral)
  3. Nonviral infection: tuberculosis and suppurative
  4. Intrapericardial hemorrhage
  5. Wounds: CT surgery
  6. Uremia
  7. Radiation (mediastinal)
  8. Vasculitis

Cardiac Tamponade *(cont'd.)*

- ***Clinical Manifestations:***
  1. Dyspnea, orthopnea, and fatigue
  2. ***Pulsus paradoxus***
  3. ***Neck vein distention with clear lung fields***
  4. Shock
  5. ***Decreased, muffled heart sounds***
- ***Diagnosis:***
  1. Echocardiography and cardiac catheterization
- ***Treatment:***
  1. Pericardiocentesis
  2. Subxiphoid surgical drainage

Constrictive Pericarditis *(cont'd.)*

- ***Etiology:***
  1. Idiopathic
  2. Secondary to open heart surgery or thoracic radiation
  3. Postviral infection
- ***Clinical Manifestations:***
  1. Dyspnea on exertion (common)
  2. Orthopnea
  3. S/S of right sided-failure
  4. Kussmaul sign
  5. Distant heart sounds and a "pericardial knock" (often confused with S_3)

Constrictive Pericarditis *(cont'd.)*

- ***Diagnosis:***
  1. ECG → low voltage and nonspecific T-wave changes
  2. Chest x-ray → normal
  3. Chest CT or MRI → thickened pericardium, pericardial calcifications if tuberculous

- ***Differential Diagnosis:***
  1. Restrictive cardiomyopathy

Constrictive Pericarditis *(cont'd.)*

- ***Treatment:***
  1. Mild sodium restriction
  2. Diuretics
  3. Pericardiectomy if required

KAPLAN

MEDICAL

Rate and Rhythm Disturbances

Sinus Bradycardia

- ***Ventricular complexes of normal width, spacing, and rate <60 beats per minute***
- ***Etiology:***
  1. Excess vagal tone: acute MI, carotid sinus pressure, vomiting, Valsalva maneuver, phenothiazines, digitalis
  2. Decreased SA node automaticity: β-blockers, CCAs
  3. Marathon running and swimming
  4. Hypothyroidism
  5. Normal variants

Sinus Bradycardia *(cont'd.)*

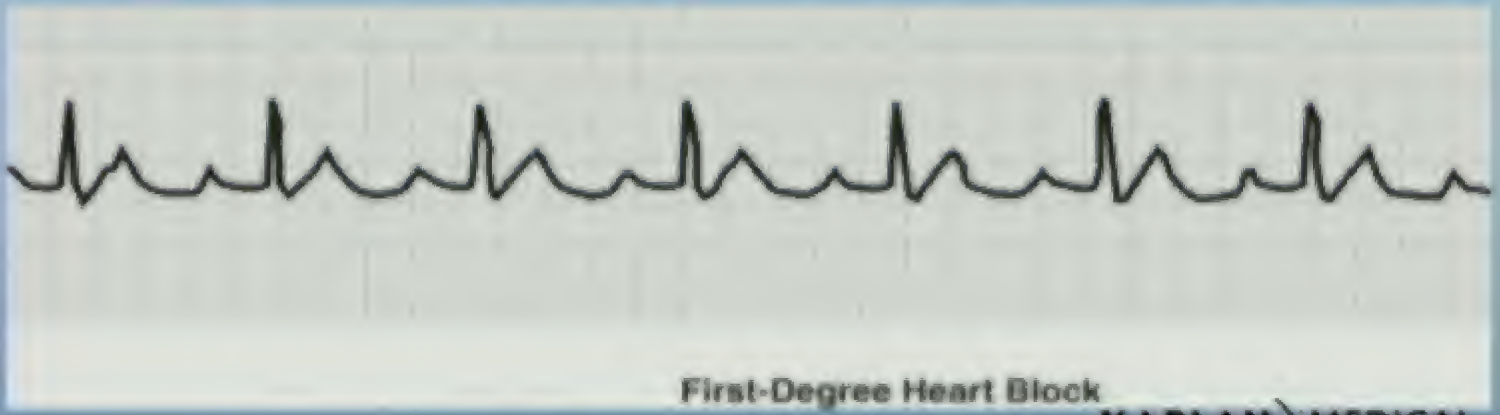
- ***Treatment:***
  1. No symptoms = no treatment
  2. Acute symptoms → atropine
  3. Chronic symptoms and bradycardia → pacemaker

Atrioventricular (AV) Block

- ***Clinical, based on ECG:***
  1. First-degree block
  2. Second-degree block
  3. Third-degree block

First-Degree Block

- PR interval >0.20 seconds
- Etiology: aging, digitalis, increased vagal tone, ischemia, inflammation, cardiomyopathy



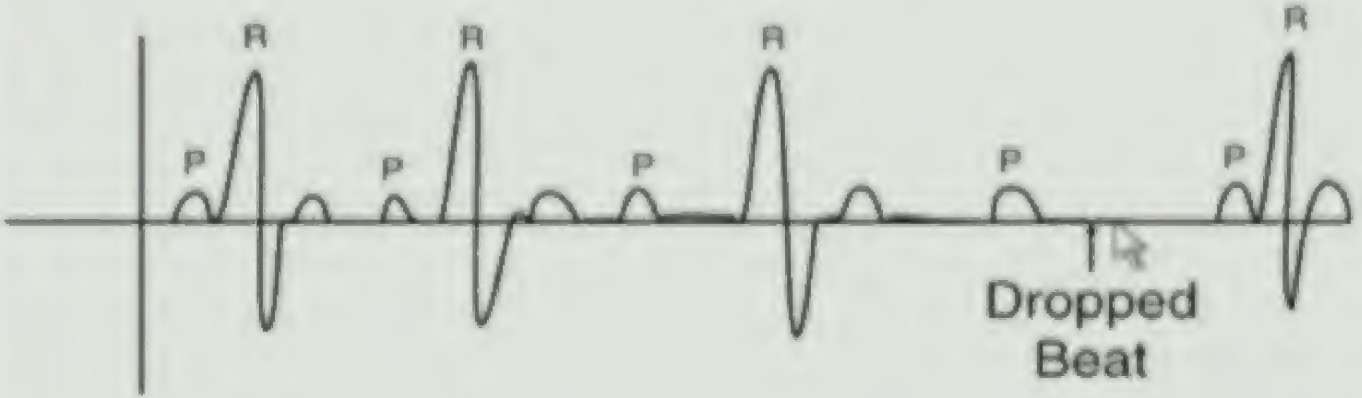
First-Degree Heart Block

KAPLAN MEDICAL

Second-Degree Block

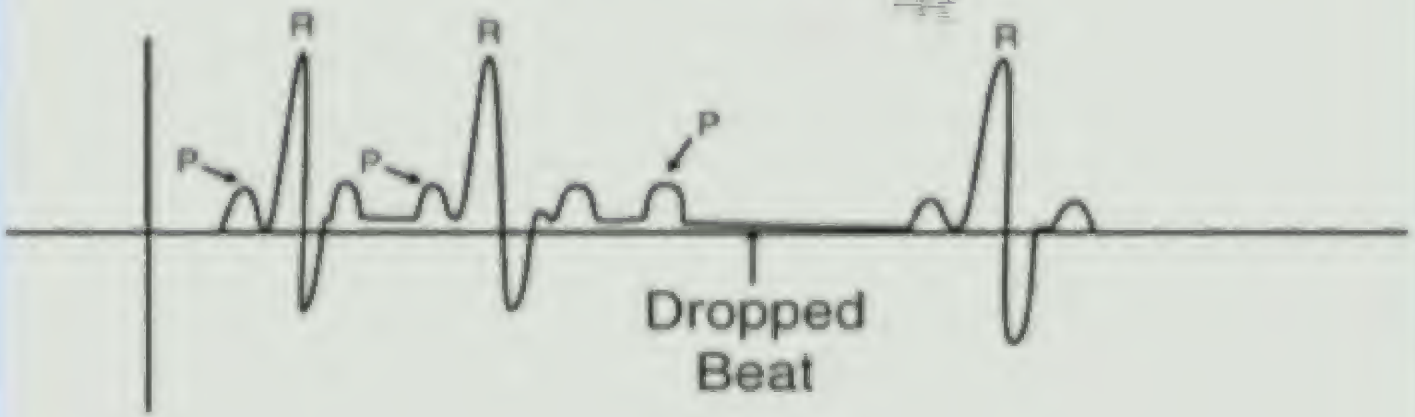
	Mobitz Type I (Wenckebach)	***Mobitz Type II***
Site of block	AV node	Infranodal
ECG	PR interval progressively lengthens until beat is "dropped"	PR interval usually of normal duration and consistent

Mobitz Type I



KAPLAN MEDICAL

Mobitz Type II



KAPLAN MEDICAL

Third-Degree Block

- **Blockage of all atrial beats;** ventricles beat through an escape focus distal to the block

Complete Heart Block

KAPLAN MEDICAL

Third-Degree Block *(cont'd.)*

- ***Clinical Manifestations:***
  1. Adams-Stoke attacks +/- CHF
  2. What is an Adams-Stoke attack???

- ***Treatment:***
  1. Pacing

Sinus Tachycardia

- Ventricular complexes are of normal width, evenly spaced, and rate is >100 BPM
- ***Etiology:***
  1. Secondary to any condition that increases the heart rate:
     - Fever
     - Hypotension
     - Pain
     - Anxiety
     - Drugs
     - Thyrotoxicosis

Paroxysmal Supraventricular Tachycardia

- Sudden onset of an ectopic tachyarrhythmia followed by abrupt cessation
- Initiated by a supraventricular premature beat
- Includes paroxysmal atrial tachycardia
- 80% due to re-entry (AV node)
- Rate between 130-200 beats/minute and regular

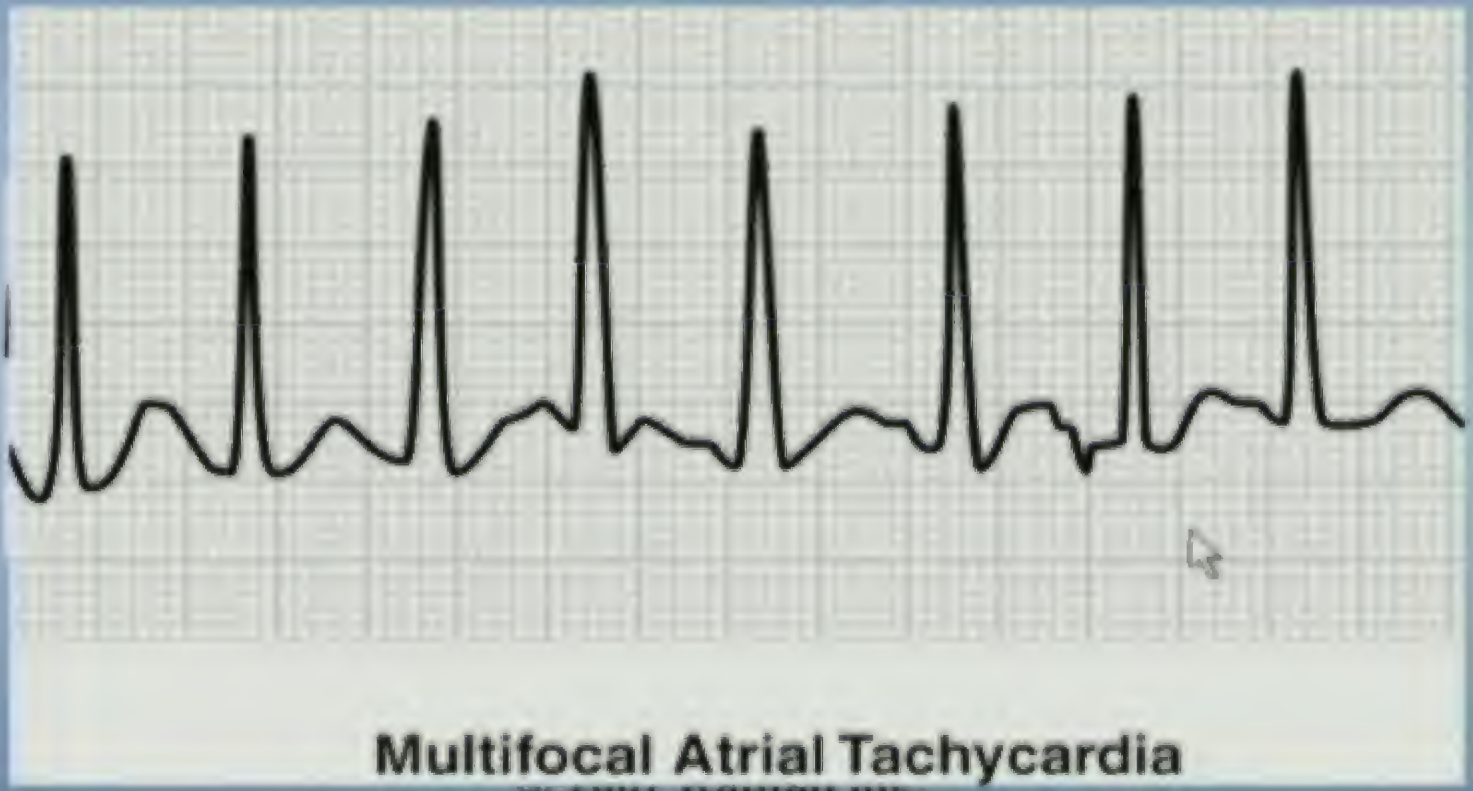
Paroxysmal Supraventricular Tachycardia *(cont'd.)*

- ***Treatment:***
  1. Increase vagal tone → carotid sinus massage
  2. Drugs of choice → ***IV verapamil or adenosine***
     - Cures 90% of cases
  3. IV propanolol or esmolol
  4. IV digitalis

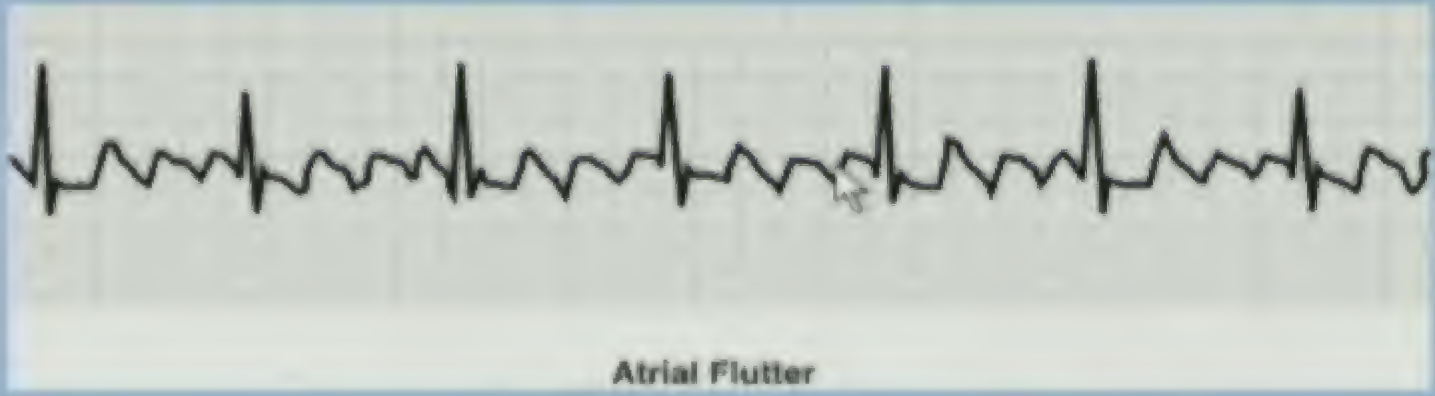
Multifocal Atrial Tachycardia

- Irregular, supraventricular rhythm between 100-200 beats/minute
- P-wave morphology and PR-interval ***varies from beat to beat*** (at least 3 to be diagnostic)
- Each ***QRS*** complex is ***preceeded*** by a ***P-wave***
- Seen in the elderly and those with respiratory failure

Multifocal Atrial Tachycardia
(cont'd.)



Atrial Flutter *(cont'd.)*



Atrial Flutter

KAPLAN MEDICAL

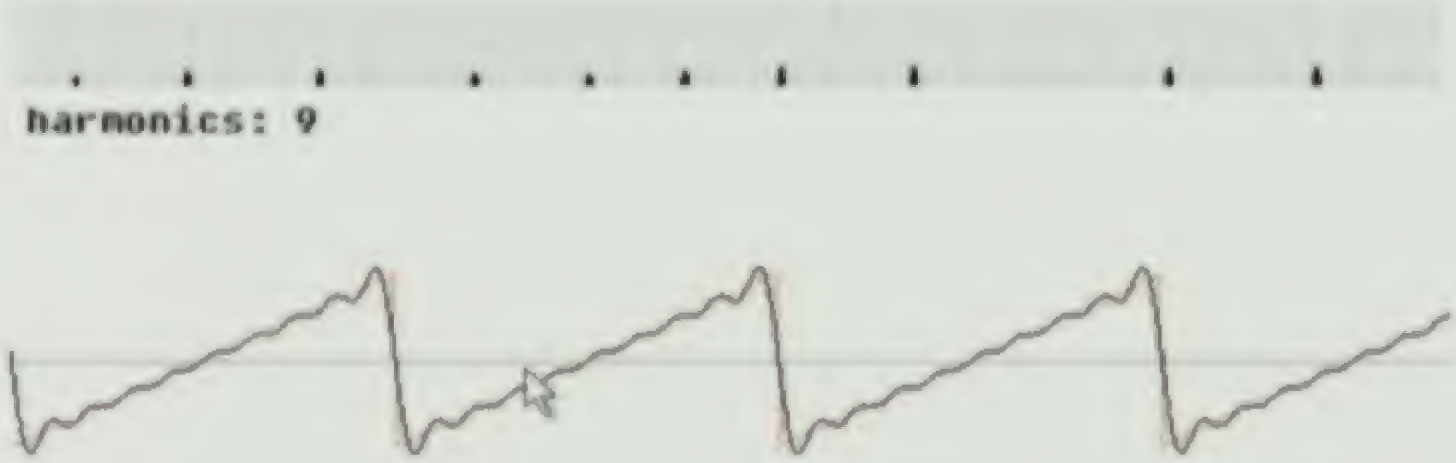
Atrial Flutter *(cont'd.)*

- ***Treatment:***
  1. Cardioversion if hemodynamically unstable
  2. Digitalis
  3. Verapamil, diltiazem
  4. β-blockers

Atrial Fibrillation

- ***Etiology/Pathophysiology:***
  1. Supraventricular tachyarrhythmia
  2. Uncoordinated atrial activation
  3. Decline in atrial function
  4. P-waves are replaced by fibrillatory waves
  5. Rapid, irregular ventricular response occurs but is dependant on several factors:
     - Electrophysiology of the AV node
     - Level of sympathetic and parasympathetic tone

Atrial Fibrillation *(cont'd.)*

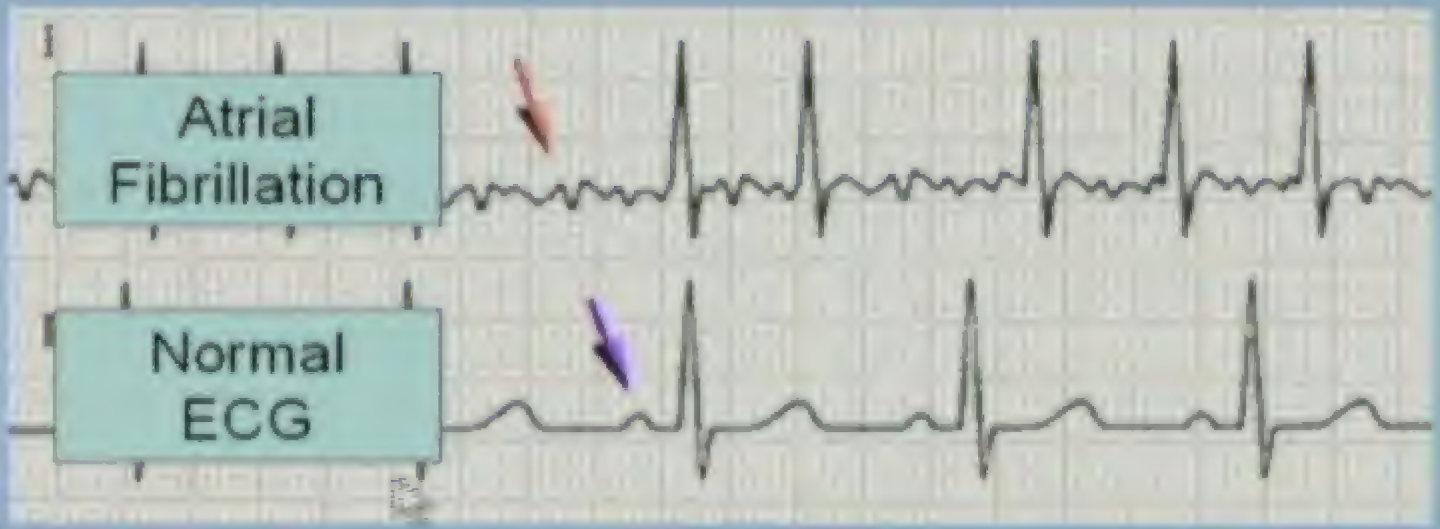


KAPLAN MEDICAL

Atrial Fibrillation *(cont'd.)*

- ***Evaluation of patients with A-fib:***
  1. Is this the first episode?
  2. Is the patient symptomatic?
  3. Is the disease self-limited?
- ***Regardless of symptomatology, the risk of a thromboembolic event is equal, whether symptomatic or not, if the A-fib persists!!!***

Atrial Fibrillation *(cont'd.)*



This image was reproduced from Wikipedia, http://www.wikipedia.com

Atrial Fibrillation *(cont'd.)*

- ***Management of A-fib:***
  1. Rhythm control versus rate control
     - RACE trial and AFFRIM trial
  2. In general:
     - Rate control is applied to the asymptomatic or those with little discomfort.
     - Rhythm control is applied to those with immediate symptoms or those who are unstable.

Atrial Fibrillation *(cont'd.)*

- ***Ventricular Rate Control:***
  1. Goal is <100 bpm
  2. Does not convert the rhythm but aids to stabilize the patient acutely
  3. β-blockers → atenolol, metoprolol
  4. CCAs → verapamil, diltiazem
  5. Beta blockers or Digoxin are the drugs of choice in those with CHF

Atrial Fibrillation *(cont'd.)*

- ***Rhythm Control:***
  1. ***Mechanical Cardioversion:***
     - Synchronized electric shock
     - Can be performed electively or emergently
     - If performing electively → ***anticoagulate the patient!!!***

Atrial Fibrillation *(cont'd.)*

- ***Rhythm Control* (cont'd.):**
  2. ***Pharmacologic Cardioversion:***
     - Less effective than synchronized cardioversion
     - Anticoagulation
     - Drugs: Amiodarone, dofetilide, flecainide, ibutilide, propafenone, and quinidine
       - ***Drugs to maintain sinus rhythm:***
         - » Amiodarone, disopyramide, dofetilide, flecainide, propafenone, and sotalol

Atrial Fibrillation *(cont'd.)*

- ***Newer treatment techniques:***
  1. Catheter ablation
     - Foci from pulmonary veins most common
     - Called the Maze procedure
- ***Other key points about A-fib:***
  1. Rate control and chronic anticoagulation is the recommended treatment for all patients with chronic A-fib.

Clinical Cases

A 32-year-old male presents with shortness of breath. His BP in the emergency room is 80/55 mm Hg. He has JVD and bilateral crackles upon physical examination. His ECG shows evidence of A-fib. What is the next best step in the management of this patient?

Electric cardioversion

WPW *(cont'd.)*

- ***Management:***
  1. Acute:
     - Unstable patient → immediate synchronized cardioversion
     - Stable patient → procainamide (drug of choice)
       - Avoid digoxin, β-blockers, and CCAs

WPW *(cont'd.)*

- ***Management:***
  1. Acute:
     - Unstable patient → immediate synchronized cardioversion
     - Stable patient → procainamide (drug of choice)
       - Avoid digoxin, β-blockers, and CCAs
  2. Long-term:
     - Ablation

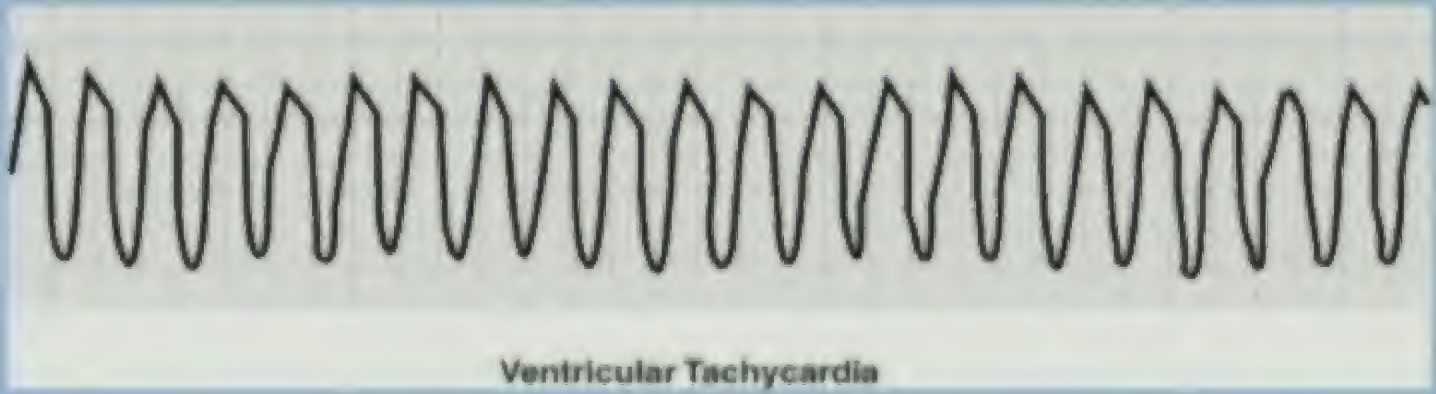
Ventricular Tachycardia (V-tach)

Defined as three or more consecutive ventricular beats at >120 bpm

Wide, bizarre QRS complexes

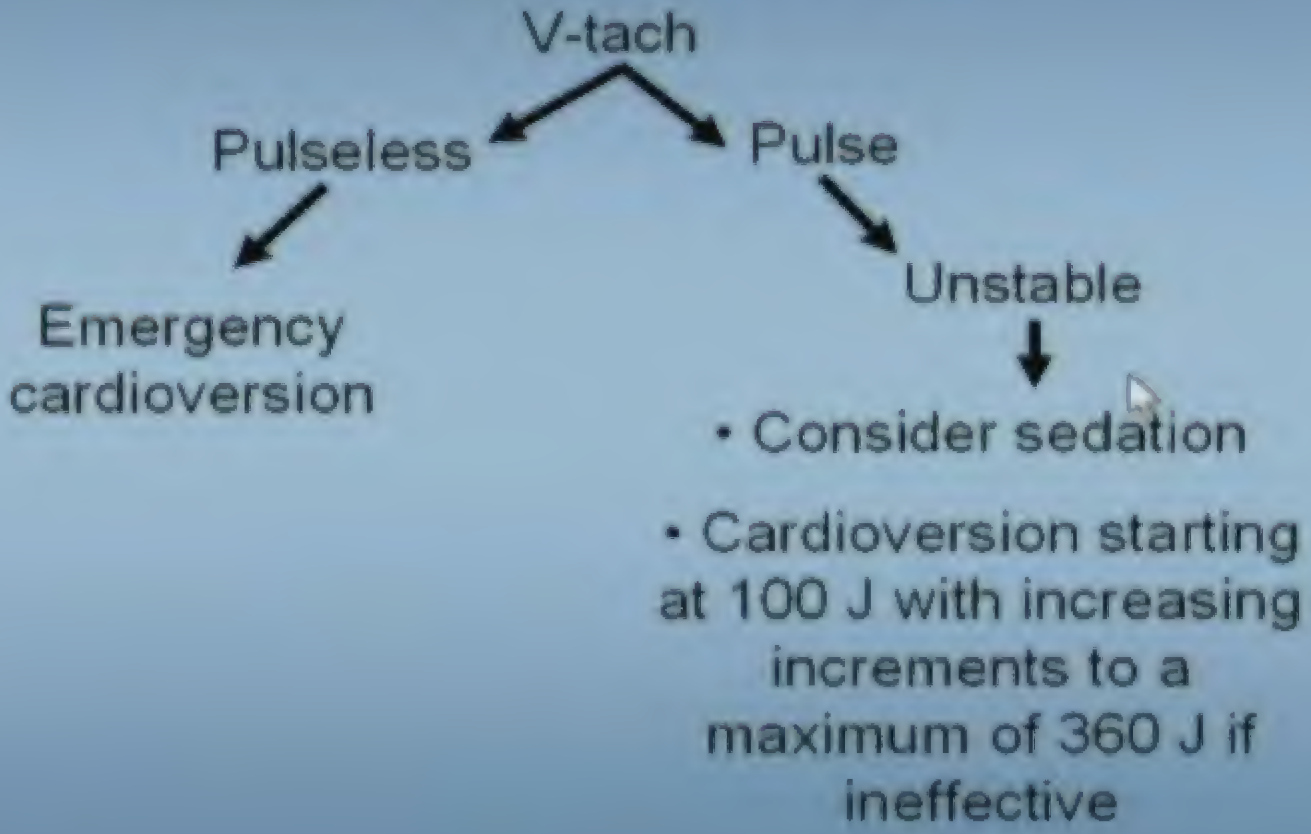
- ***Etiology:***
  1. Ischemic heart disease, especially post-MI
  2. Cardiomyopathies
  3. Metabolic derangements

V-tach *(cont'd.)*



Ventricular Tachycardia

Management of V-tach



Management of V-tach *(cont'd.)*

V-tach

↓

Pulse and hemodynamically stable

↓

- Oxygen
- Amiodarone or lidocaine 1 mg/kg loading dose

↓

Lidocaine 0.5-3 mg/kg q 8 minutes until resolution

↓

Procainamide 20-100 mg/kg until resolution

↓

Torsades de Pointes

- Undulating rotations of the QRS complex around the ECG baseline
  1. Premature ventricular beat
  2. Abnormal ventricular repolarization
  3. Prolonged QT-interval

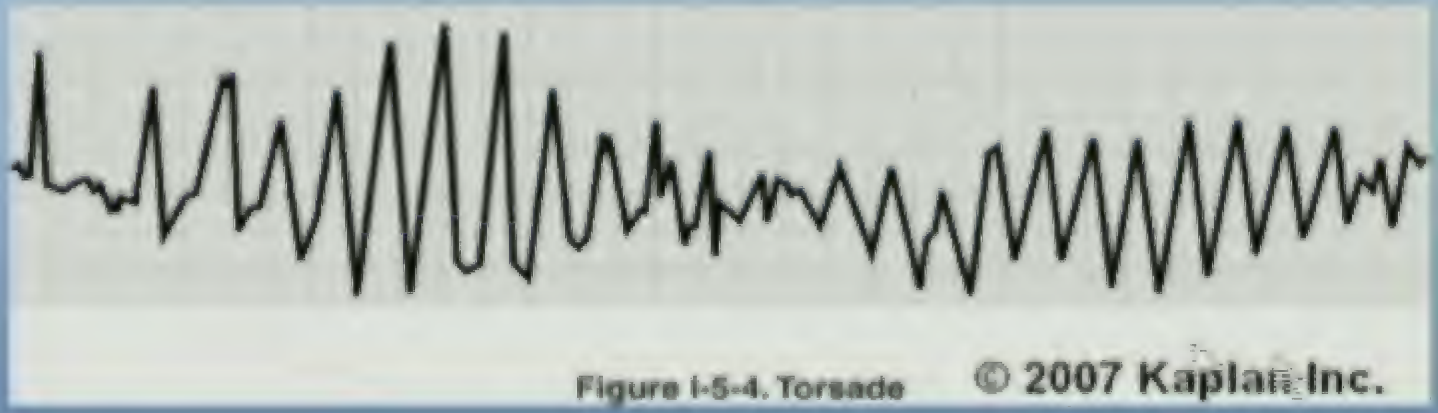


Figure I-5-4. Torsade